

Mitral valve replacement in children

Comparative study of pre- and postoperative haemodynamics and left ventricular function

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SUMMARY Haemodynamic variables and left ventricular function were studied before and after mitral valve replacement in 44 children age 3 to 17 years (mean 11.9 years). Thirty-nine Starr-Edwards prostheses and five Hancock prostheses were used; postoperative study took place two to six months (mean 3.9 months) after operation. Pulmonary hypertension was present preoperatively in most patients, with mean pulmonary artery pressures of 18 to 75 (mean 46.5 mmHg). Postoperatively there was a pronounced drop in pressure to a mean value of 25.6 mmHg, partially explained by a decrease in pulmonary capillary wedge pressure. Pulmonary arteriolar resistance, however, also decreased conspicuously from an average of $590 \text{ dynes s cm}^{-5} \text{ m}^{-2}$ preoperatively to $282 \text{ dynes s cm}^{-5} \text{ m}^{-2}$ postoperatively. A return to normal resistance was seen in every case when preoperative resistance did not exceed $650 \text{ dynes s cm}^{-5} \text{ m}^{-2}$; above this threshold some degree of pulmonary hypertension often persisted. The residual gradient across the prosthetic valve was slightly higher for the Hancock than for the Starr-Edwards prosthesis (mean 8.7 mmHg, vs mean 6.9 mmHg).

The left ventricular end-diastolic volume was much increased before surgery, with a mean value of 190 ml/m^2 ; it decreased conspicuously after operation to 103 ml/m^2 . The left ventricular ejection fraction ranged from 40% to 76% (mean 57%) before operation; there was no significant change after operation, with values ranging from 40% to 73%.

This left ventricular dysfunction is probably the result of myocardial injury caused by a chronic volume overload and the sequelae of rheumatic carditis.

Mitral valve disease with regurgitation and stenosis gives rise to various haemodynamic consequences. First, there is left ventricular volume overload and dilatation of the left atrium and ventricle. Longstanding left ventricular volume overload may compromise left ventricular function.^{1 2} If the mitral valve disease is of rheumatic origin, left ventricular function may also be depressed as a consequence of myocardial fibrosis, secondary to myocarditis. Secondly, pulmonary venous congestion will result in pulmonary hypertension; with time, pulmonary vascular disease may develop, compromising the return to normal pressures after mitral valve function is restored. Severe mitral valve disease often requires valve replacement, which is usually reserved for very disabled patients. Various degrees of left ventricular dys-

function and of pulmonary hypertension are usually therefore present, and the reversibility of such changes is uncertain. Haemodynamic assessment after mitral valve replacement is of use and has often been performed in adult patients,³⁻⁵ but very few data are available in children.⁶ We did not find any data concerning left ventricular function and volume after valve replacement in children.

We would like to report the results of postoperative catheterisation in 44 children who have undergone valve replacement for severe mitral valve disease, including the results of left ventricular volume studies in 30 children.

Patients and methods

Forty-four children (23 boys and 21 girls) with clinical and haemodynamic evidence of severe mitral valve

disease were studied. The valve lesion was the result of rheumatic heart disease in 35, and of congenital malformation in nine. Their ages ranged from 3 to 17 years (mean 11.9 years). Twenty-nine patients had mitral regurgitation associated with mitral stenosis; 15 patients had pure mitral regurgitation. In 30 patients, there was significant disease of another valve: 12 had aortic regurgitation, 12 had tricuspid regurgitation, six had aortic and tricuspid regurgitation. Two had a ventricular septal defect. Thirty-three patients were in grade IV heart failure (according to the New York Heart Association), nine in grade III, and two in grade II. Thirty-nine underwent mitral valve replacement with a Starr-Edwards prosthesis and five with a Hancock prosthesis; nine patients also had a tricuspid annuloplasty (De Vega), and two aortic valve replacement (one Björk and one Starr-Edwards prosthesis). Two patients had closure of a ventricular septal defect.

These 44 patients are part of a group of 171 children who underwent valve replacement from 1969 to 1980 in Geneva.⁷ Postoperative catheterisation was performed either to assess pulmonary pressure (when preoperative values were raised) or to rule out valve dysfunction in the presence of a systolic or diastolic murmur.

Cardiac catheterisation was performed two to six months (mean 3.9 months) after operation. The procedure was done under light sedation with a mixture of pethidine, promethazine, and chlorpromazine.

Routine cardiac catheterisation was performed by percutaneous puncture of the right femoral artery and vein. Cardiac output was measured by the Fick method, with oxygen consumption assumed (table of La Farge and Miettinen). Pulmonary vascular resistance was calculated by the following formulae: $TPVR = PA \times 80 / CI$; $PAR = (PA - PAW) \times 80 / CI$, where $TPVR$ is the total pulmonary vascular resistance in dynes $s\ cm^{-5}$, PAR is the pulmonary arteriolar resistance, PA and PAW are the mean pulmonary arterial and pulmonary arterial wedge pressures (mmHg), respectively, and CI is the cardiac index (litres/min per m^2).

Thirty patients underwent left ventricular cineangiography in the right anterior oblique projection using 1 to 1.5 ml/kg of 76% Urografin. Ventricular volumes were calculated using the Sandler and Dodge formula⁸ from planimetered surfaces on the right anterior oblique cineangiogram. Ejection fraction (EF) was calculated with the following formula: $EF = (EDV - ESV) / EDV \times 100$, where EDV and ESV are end-diastolic and end-systolic volumes, respectively. We took cardiac cycles early after opacification to obtain physiological information from cineangiography.⁹ The pre- and postextrasystolic systoles were excluded. Mean frequency systoles were taken when the patient was in atrial fibrillation. Absolute and indexed volume is reported in 27 patients only because the magnification factor was unknown in three.

Table Haemodynamics and left ventricular function data

Case no.	Sex	Age		PA	PAW	MG	CI	TPVR	PAR	NYHA	RMR	Surgical treatment	LVED	EF	LVES
1	F	15	Preop	45/24	33	21	2.9	910	333	IV		Starr M	80	60	31
			Postop	35/18	25	15	8	500	200	I-II	-	An. T	50	56	22
2	M	15	Preop	90/50	68	30	—	2190	1220	IV		Starr M	—	—	—
			Postop	28/13	20	7	8	330	212	I-II	-		—	—	—
3	M	13	Preop	40/20	28	20	2.5	510	150	IV		Starr M	379	61	147
			Postop	28/11	16	13	7	320	60	I	-		189	53	89
4	F	11	Preop	65/45	55	30	—	2380	1050	IV		Starr M	—	—	—
			Postop	36/10	17	6	5	350	240	II	-		—	—	—
5	F	13	Preop	42/13	34	28	17	1130	200	IV		Starr M	176	62	67
			Postop	26/3	16	8	7	310	160	I-II	-		109	50	56
6	M	7	Preop	30/10	24	13	—	480	220	III		Hancock M	—	—	—
			Postop	25/13	17	8	6	290	150	I-II	-		—	—	—
7	F	13	Preop	105/58	75	45	44	3160	1260	IV		Starr M	337	65	120
			Postop	40/14	26	5	2	520	420	I-II	-		119	65	42
8	F	16	Preop	58/29	38	28	2.2	1220	920	IV		Starr M	95	64	34
			Postop	30/12	20	11	10	470	210	I-II	-		59	52	29
9	M	17	Preop	80/40	60	29	2.7	1780	920	IV		Starr M	116	54	54
			Postop	75/40	50	15	8	1900	1330	II	+		136	41	80
10	F	16	Preop	58/25	40	30	—	1450	360	IV		Starr M	196	76	46
			Postop	25/14	18	11	8	580	220	I-II	-		85	48	45
11	M	5	Preop	45/20	30	15	—	890	440	IV		Starr M	—	—	—
			Postop	29/16	18	7	?	390	190	I-II	-		—	—	—
12	F	10	Preop	38/28	32	24	18	1510	380	IV		Starr M	94	49	48
			Postop	32/12	24	15	6	600	225	I-II	-		92	44	52
13	M	5	Preop	50/35	42	21	5	1680	840	IV		Starr M	—	59	—
			Postop	48/26	35	16	10	1000	540	—	+	An. T	—	47	—
14	M	14	Preop	85/48	59	30	10	2250	1100	IV		Starr M	197	55	89
			Postop	70/38	50	19	10	1740	1080	—	+	An. T	183	50	91

Table (cont) Haemodynamics and left ventricular function data

Case No.	Sex	Age		PA	PAW	MG	CI	TPVR	PAR	NYHA	RMR	Surgical treatment	LVED	EF	LVES
15	M	17	Preop	65/25 39	19	10	—	—	—	IV	—	Starr M	218	50	109
			Postop	28/12 16	8	5	—	—	—	I-II	—	VSD clos.	—	47	—
16	M	10	Preop	82/35 55	24	12	2.5	1760	990	IV	—	Starr M	159	58	67
			Postop	38/13 23	14	8	3.6	510	200	I	—	An. T	114	74	30
17	F	11	Preop	46/26 34	14	—	2.5	1090	640	IV	—	Starr M	118	58	50
			Postop	36/14 25	13	8	3.8	530	253	I	—	—	87	62	33
18	F	12	Preop	26/12 18	9	7	3.8	380	190	III	—	Starr M	—	—	—
			Postop	28/14 17	11	?	4.8	280	100	I-II	—	—	—	—	—
19	M	13	Preop	90/56 66	28	20	2.7	1960	1130	IV	—	Starr M	—	—	—
			Postop	24/8 13	6	?	3.6	290	150	I	—	—	—	—	—
20	F	14	Preop	85/40 60	28	—	2.0	2400	1280	IV	—	Starr M	—	—	—
			Postop	37/24 27	9	8	3.8	570	380	I	—	—	—	—	—
21	M	15	Preop	84/42 60	20	20	—	—	—	III	—	Starr M	—	—	—
			Postop	36/18 25	11	8	—	—	—	I	—	VSD clos.	—	—	—
22	M	10	Preop	96/53 72	16	—	3.9	1480	1150	IV	—	Starr M	—	—	—
			Postop	34/15 24	12	?	4.5	430	210	II	—	—	—	—	—
23	F	—	Preop	28/12 16	12	—	—	—	—	III	—	Starr M	—	—	—
			Postop	29/10 18	13	?	—	—	—	II	—	—	—	—	—
24	F	7	Preop	90/50 68	30	24	4.2	1300	720	IV	—	Starr M	292	52	134
			Postop	32/15 20	6	3	5.9	270	190	I	—	—	80	56	35
25	M	11	Preop	37/16 24	20	7	3.0	640	110	III	—	Starr M	361	68	115
			Postop	26/10 14	9	7	3.4	330	120	I	—	—	60	70	19
26	F	11	Preop	45/20 30	18	10	2.8	860	340	II	—	Starr M	181	68	58
			Postop	34/16 20	12	6	4.2	380	150	I	—	—	87	66	30
27	M	15	Preop	75/35 53	37	28	2.9	1460	440	IV	—	Hancock M	68	51	33
			Postop	54/19 33	16	8.5	2.7	980	500	II	+	—	82	52	40
28	F	17	Preop	91/45 61	47	40	1.5	3250	750	IV	—	Starr M	242	41	143
			Postop	50/22 29	15	9	4.0	580	280	I-II	—	An. T	79	48	41
29	M	11	Preop	73/44 58	31	23	2.7	1720	800	IV	—	Hancock M	—	76	—
			Postop	28/14 18	14	13	5.3	270	60	I	—	—	—	53	—
30	F	6	Preop	61/33 48	26	—	2.7	1420	650	III	—	Starr M	135	49	69
			Postop	32/15 20	15	?	4.0	400	100	I	—	—	98	56	42
31	F	11	Preop	42/31 36	—	—	2.3	1250	—	IV	—	Starr M	—	—	—
			Postop	37/15 25	10	?	3.2	630	380	I-II	—	—	—	—	—
32	M	13	Preop	70/35 50	27	18	2.7	1480	680	IV	—	Starr M	113	49	58
			Postop	44/15 27	12	?	3.2	680	370	I-II	—	—	108	61	42
33	F	13	Preop	45/28 36	23	8	3.3	870	320	III	—	Starr M	110	64	40
			Postop	24/10 15	6	6	3.5	340	210	I-II	—	—	87	63	32
34	M	14	Preop	60/33 48	30	10	2.5	1540	580	III	—	Starr M	257	55	117
			Postop	31/11 18	9	6	3.4	420	210	I-II	—	Starr A	129	68	40
35	M	13	Preop	96/60 73	32	30	3.0	1950	1090	IV	—	Starr M	171	62	65
			Postop	34/12 23	5	4.5	3.6	510	400	I	—	—	55	57	23
36	M	10	Preop	80/45 58	30	25	2.8	1660	800	IV	—	Starr M	255	45	140
			Postop	41/25 31	14	7	3.5	710	390	II	+	—	250	47	133
37	M	3	Preop	55/29 41	26	16	2.0	1640	600	IV	—	Starr M	—	—	—
			Postop	28/13 20	12	5	4.0	400	160	I-II	—	—	—	—	—
38	M	16	Preop	57/38 45	30	23	2.0	1800	600	IV	—	Hancock M	82	43	47
			Postop	37/17 22	12	7	3.2	550	250	I-II	—	An. T	65	54	30
39	M	15	Preop	100/40 60	24	—	2.6	1850	1110	IV	—	Starr M	—	—	—
			Postop	38/17 27	12	8	4.4	490	270	I-II	—	An. T	—	—	—
40	F	13	Preop	110/40 60	31	15	2.6	1850	890	IV	—	Starr M	188	64	68
			Postop	50/23 33	16	10	4.7	560	290	II	—	An. T	63	73	17
41	M	10	Preop	46/23 32	21	—	2.6	990	340	III	—	Hancock M	191	60	77
			Postop	37/18 25	15	9	3.5	570	230	I	—	—	84	40	51*
42	F	10	Preop	30/17 22	11	—	3.6	480	240	II	—	Starr M	—	—	—
			Postop	29/13 17	13	?	3.6	380	90	I	—	Björk A	—	—	—
43	F	13	Preop	77/22 44	26	—	3.5	1010	410	IV	—	Starr M	241	54	109
			Postop	28/12 19	13	5	5.0	300	100	I	—	—	114	55	51
44	F	11	Preop	48/20 30	20	12	2.4	990	330	IV	—	Starr M	200	55	89
			Postop	28/10 18	7	6	3.8	380	180	I	—	—	72	50	35
Mean			Preop	46.5	25.6	—	2.6	1340	590	—	—	—	190	57	—
Mean			Postop	23.4	10.9	—	3.8	533	282	—	—	—	103	56	—
p				<0.0001	<0.0001	—	<0.0001	<0.0001	<0.0001	—	—	—	<0.0001	>0.15	—

A, aortic; An.T, tricuspid annuloplasty (De Vega); CI, cardiac index (l/min per m²); EF, ejection fraction (%); LVED, left ventricular end-diastolic volume (ml/m²); LVES, left ventricular end-systolic volume (ml/m²); M, mitral; MG, mitral gradient (mmHg); NYHA, New York Heart Association grade; PA, pulmonary artery pressure (mmHg); PAR, pulmonary arteriolar resistance (dynes s cm⁻⁵ m⁻²); PAW, pulmonary arterial wedge pressure (mmHg); RMR, residual mitral regurgitation; TPVR, total pulmonary vascular resistance (dynes s cm⁻⁵ m⁻²); VSD clos., ventricular septal defect closure.

Results

Complete data are shown in the Table.

PRESSURES

Pulmonary artery pressure was often much increased before operation, up to systemic level in some cases. The mean pulmonary artery pressure preoperatively ranged from 18 to 75, mean 46.5 mmHg. After operation, there was a pronounced decrease to values ranging from 10 to 42, mean 23.4 mmHg. This was highly significant ($p < 0.0001$). In five patients, however, there was mitral regurgitation postoperatively, and pulmonary arterial pressure remained high (above 30 mmHg) (Fig. 1a).

Mean pulmonary arterial wedge pressure was high before operation (9 to 45, mean 25.6 mmHg); it decreased after operation (5 to 19, mean 10.6 mmHg)

($p < 0.0001$). Five patients had pulmonary arterial wedge pressures higher than 15 mmHg after operation; each had mitral regurgitation caused by paravalvar leak (Fig. 1b).

CARDIAC INDEX

The cardiac index rose from an average of 2.6 litres/min per m^2 before operation, to 3.8 litres/min per m^2 ($p < 0.0001$) after operation (lower limit of normal for our laboratory is 3.0 l/min per m^2). In patients with residual mitral regurgitation, cardiac index was lower than 3.5 litres/min per m^2 postoperatively (Fig. 2).

RESISTANCES

Total pulmonary vascular resistance was moderately or much raised before operation (450 to 3150, mean 1340 dynes $s\ cm^{-5}\ m^{-2}$), and it decreased after operation (250 to 970, mean 533) ($p < 0.0001$).

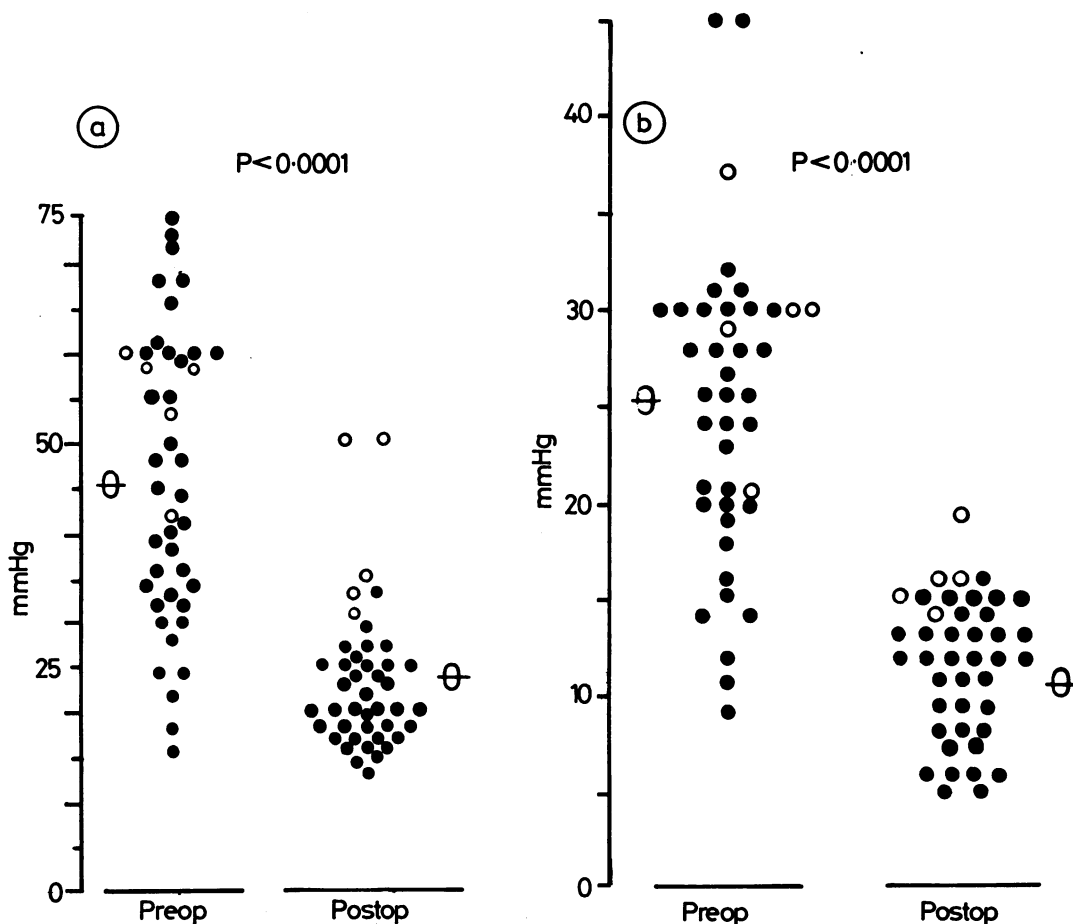


Fig. 1 (a) Mean pulmonary artery pressure before and after mitral valve replacement. Open circles indicate patients with residual mitral regurgitation (paravalvar leak). (b) Mean pulmonary wedge pressure before and after mitral valve replacement. Open circles indicated patients with residual mitral regurgitation.

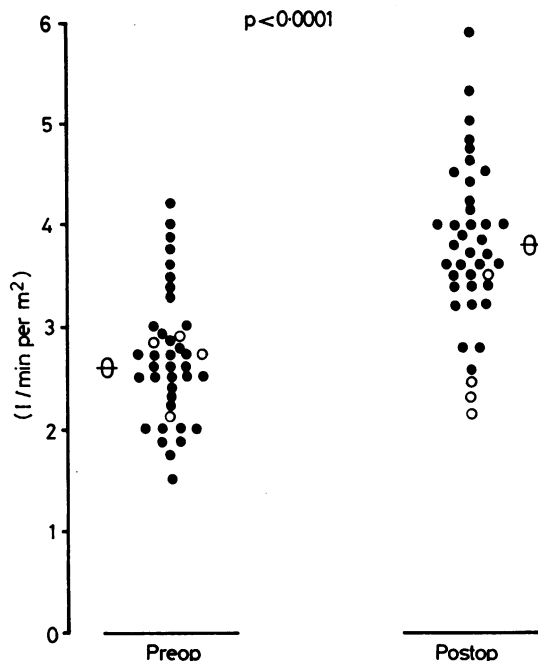


Fig. 2 Cardiac index before and after mitral valve replacement. Open circles indicate patients with residual mitral regurgitation.

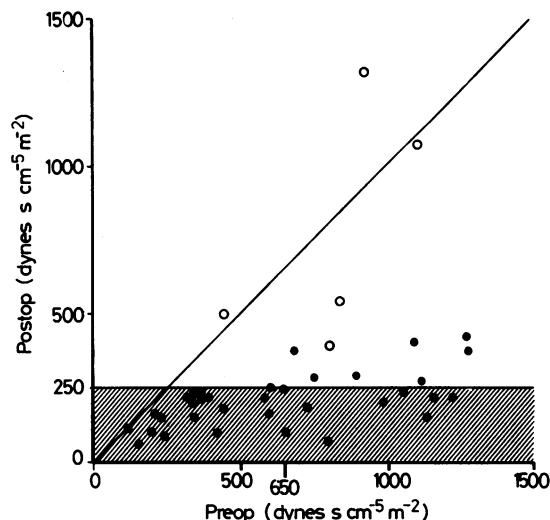


Fig. 3 Postoperative pulmonary arteriolar resistance (on the y axis) plotted against preoperative resistance (on the x axis), with the line of identity. Shaded area indicates normal values. Open circles are patients with residual mitral regurgitation. In cases without residual regurgitation, resistances always return to normal when the preoperative value does not exceed 650 dynes $s\ cm^{-5}\ m^{-2}$.

Pulmonary arteriolar resistance showed values between 120 and 1280, mean 590 dynes $s\ cm^{-5}\ m^{-2}$ before operation; these values fell to 60 to 430, mean 282 dynes $s\ cm^{-5}\ m^{-2}$ after operation ($p < 0.0001$) (Fig. 3). In all patients with preoperative values lower than 650 dynes $s\ cm^{-5}\ m^{-2}$, postoperative resistance was found to be normal (≤ 250 dynes $s\ cm^{-5}\ m^{-2}$). If preoperative values exceeded 650 dynes $s\ cm^{-5}\ m^{-2}$, the pulmonary arteriolar resistance decreased, but a return to normal was not the rule. Patients with residual regurgitation had pulmonary arteriolar resistance above 500 dynes $s\ cm^{-5}\ m^{-2}$, unchanged from preoperative values (Fig. 3).

RESIDUAL MITRAL GRADIENT

A residual mitral gradient was commonly found after valve replacement in children. This was mild for the Starr-Edwards prostheses, with a gradient ranging from 2 to 10, mean 6.9 mmHg. For the Hancock prostheses, the gradient was somewhat higher, between 6 and 13, mean 8.7 mmHg (not significant). Though the difference between means is not statistically significant, it appears that one third of the patients with Starr-Edwards prostheses have insignificant gradients, between 2 and 6 mmHg; no child with a Hancock prosthesis had such a small gradient.

VENTRICULAR VOLUMES

End-diastolic left ventricular volumes were raised before operation (83 to 379, mean 190 ml/ m^2). Seventeen patients had left ventricular end-diastolic volumes higher than 150 ml/ m^2 , five patients had values between 110 and 150 ml/ m^2 , but five had values lower than 100 ml/ m^2 . Left ventricular end-diastolic volumes decreased after valve replacement and showed values between 55 and 250, mean 103 ml/ m^2 ($p < 0.0001$) (Fig. 4). In 16 patients, the ventricular volume fell below 100 ml/ m^2 , and six had values between 100 and 130 ml/ m^2 . Five still had values above 130 ml/ m^2 . Among these there was a case of moderate aortic regurgitation, who needed aortic valve replacement later; in three other patients, high postoperative left ventricular volume was related to residual mitral regurgitation.

EJECTION FRACTION

Left ventricular ejection fraction was measured before and after operation (Fig. 5). Before operation, about half of the patients had a depressed ejection fraction; the values ranged from 40 to 76%, mean 57%. Thirteen patients had values higher than 60%, 10 patients had values between 50 and 60%, and seven patients had values lower than 50%. After operation, these values did not change significantly; they were

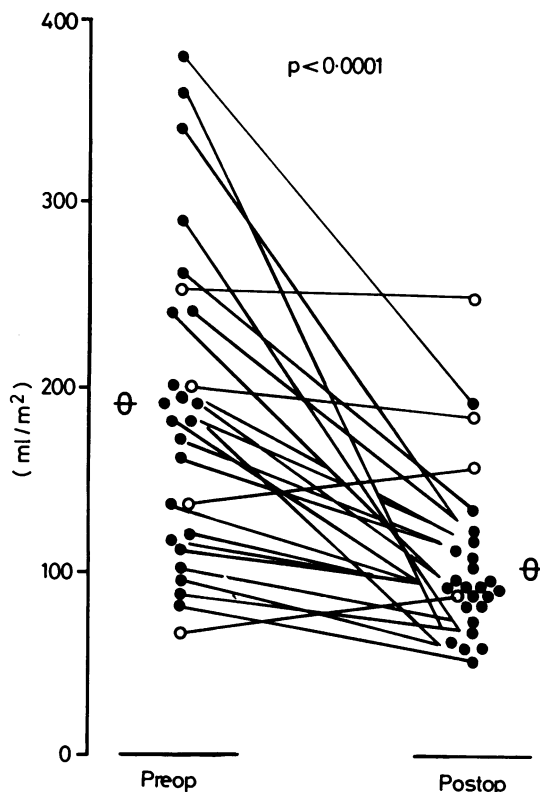


Fig. 4 Left ventricular end-diastolic volume (LVED) before and after mitral valve replacement. LVED decreased significantly after valve replacement in patients without residual mitral regurgitation. Open circles indicate patients with residual mitral regurgitation.

situated between 40 and 73%, mean 56%. No relation was found between the ejection fraction and age of the patient.

Discussion

Mitral valve replacement in children is usually restricted to patients who are severely symptomatic and in whom mitral valve repair is not feasible. The short term results are satisfactory and the children are much improved.⁷ Since valve replacement, however, is usually performed after a long period of progressive heart failure, complications are often present: long-standing left ventricular overload, together with sequelae of myocarditis in the case of rheumatic heart disease, may result in left ventricular dysfunction; on the other hand, chronically increased left atrial pressure will result in pulmonary hypertension. It is therefore interesting to assess postoperative haemodynamics and left ventricular function after valve replace-

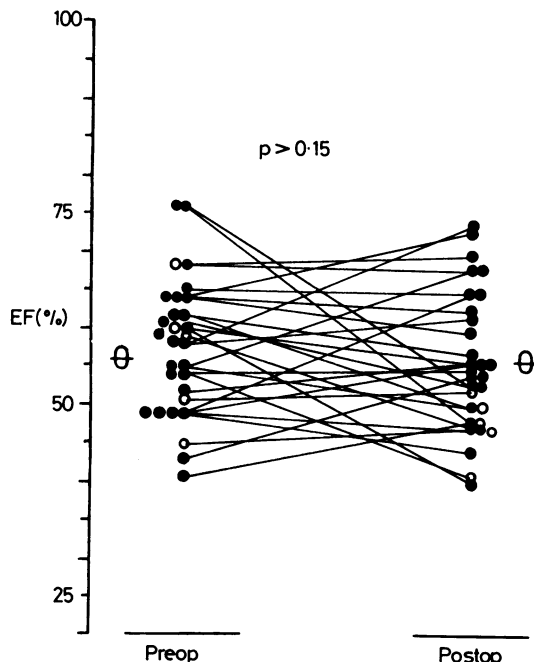


Fig. 5 Left ventricular ejection fraction (EF) measured before and after operation. The values are often below normal before as well as after operation, and there is no significant difference between preoperative and postoperative values.

ment and to compare the results with preoperative data.

HAEMODYNAMIC VARIABLES

Even extreme degrees of pulmonary hypertension and increased pulmonary vascular resistance secondary to mitral valve disease regress conspicuously in adult patients^{3, 4} and children⁶ who underwent adequate valve replacement, though a return to normal is not the rule. Thus, severe pulmonary hypertension is no longer a contraindication for operation, as had been suggested previously by some.¹⁰ This regression is partially the result of left atrial decompression: the pronounced reduction in left atrial pressure (pulmonary wedge pressure) explains the impressive reduction in total pulmonary vascular resistance. As shown in the present study, however, pulmonary arteriolar resistance, which is not directly related to left atrial pressure, also decreases conspicuously. The degree of increase in pulmonary arteriolar resistance before

operation seems to be a determinant of residual post-operative pulmonary hypertension. Indeed, when preoperative values were below $650 \text{ dynes s cm}^{-5} \text{ m}^{-2}$, a return to normal resistance was always observed. Above this level, some degree of pulmonary hypertension often persisted. In four of our seven patients with increased pulmonary arteriolar resistance after operation and no mitral regurgitation, the residual mitral gradient was somewhat above the average. In no case, however, did it exceed 10 mmHg and we do not believe that this alone would explain the increased pulmonary arteriolar resistance. As the postoperative catheterisation took place fairly soon after operation, we do not know whether a further decrease may occur later in these cases. It is obvious, however, that the high resistances measured before operation are not the result of advanced pulmonary vascular disease, but rather of pulmonary arteriolar spasm. The situation is obviously different from pulmonary hypertension secondary to left to right shunt; indeed, pulmonary arteriolar resistance, measured two to four months after closure of a ventricular septal defect with pulmonary hypertension, does not change from preoperative values (B Friedli, unpublished data).

We have noted a significant increase in cardiac output after operation from the usually low preoperative values. It also appeared that some degree of residual mitral gradient may be found in children after mitral valve replacement: in our series, this was somewhat more significant for the Hancock porcine xenograft than for the Starr-Edwards prostheses. This is because for the same prosthetic valve diameter, the valve orifice is smaller in the Hancock prosthesis than in the Starr-Edwards prosthesis. As the postoperative study was done early, calcification of the porcine xenograft¹¹ could not at that stage have been responsible for the relative stenosis.

Mitral regurgitation (paravalvar leak) was found in five patients. This relatively large number can be explained by the fact that the 44 patients catheterised in this study were selected from 171 children who had received valve prostheses during that period. Indications for recatheterisation were the presence of a residual murmur or preoperative pulmonary hypertension. It is obvious from the present study that, when significant residual mitral regurgitation is present, the haemodynamic variables remain abnormal, similar to the levels before operation.

LEFT VENTRICULAR VOLUME AND FUNCTION

To our knowledge, these variables have not been previously studied in children with valve prostheses. Left ventricular volume is considerably increased before operation, because of chronic left ventricular volume overload. Similar increases have previously been found experimentally¹² as well as in adult patients.¹³

After operation, there is a pronounced decrease in volume, but a return to normal is not the rule: the mean left ventricular volume remains raised around 100 ml/m^2 . Left ventricular ejection fractions before operation vary over a wide range, but they are quite often abnormally low. There was no significant change in the mean, after operation, though some individual values did either increase or decrease. It must be emphasised that ejection fractions before and after mitral valve replacement cannot be directly compared because of the different haemodynamic setting. Indeed a series of variables, which determine left ventricular stroke volume, also influence ejection fraction. They are preload (left ventricular end-diastolic volume), afterload (left ventricular wall stress during ejection), myocardial contractility, and heart rate. In mitral regurgitation, preload is increased and afterload decreased. This should enhance ejection fraction; it has been shown that the "unloading" of the left ventricle in mitral regurgitation may mask the effect of depressed myocardial contractility on overall pump function.¹⁴ This indicates that, in some of our patients, quite severe left ventricular dysfunction must have existed before operation, as the ejection fraction remains low in spite of the decreased afterload. One would also expect that ejection fraction decreases after mitral valve replacement, because of the increased afterload and decreased preload. This has been shown to occur in adults,^{15 16} but we did not find it in the present study. One explanation for this difference may be because left ventricular volume decreases considerably in children after valve replacement, more so than in adults. The reduction in left ventricular dimension (radius) reduces left ventricular stress, that is afterload. Thus, afterload increase after valve surgery in children may be less important than in adults.

As to the cause of the left ventricular dysfunction, before as well as after operation, two factors need to be considered: chronic volume overload may itself produce myocardial changes that are not completely reversible, as shown in the electron-microscopical studies of Papadimitriou *et al.*¹⁷ After closure of a ventricular septal defect—another cause of chronic left ventricular volume overload in children—Jarmakani *et al.*¹ showed some left ventricular dysfunction, except in those patients who were operated on very early, before two years of life. In the present study, we have not been able to show a clear effect of age or duration of disease on left ventricular ejection fraction. There is obviously another possible cause of myocardial dysfunction in patients with rheumatic heart disease, that is myocardial fibrosis secondary to rheumatic myocarditis. Indeed, many children in this study have had repeated attacks of rheumatic fever.

The indication for valve replacement in children

remains, as mentioned previously, the presence of severe, often longstanding heart failure. This is because of the complications of valve prostheses which occur in children,^{18 19} as well as in adults.²⁰ The price of this conservative approach may be the presence of irreversible myocardial damage at the time of operation. In adults the results of valve replacement in grade IV heart failure are not as good as those in grade III heart failure.²¹ Should a new valve prosthesis with low thromboembolic risk and long durability be developed, valve replacement at an earlier stage, for example in grade II, would therefore be indicated.

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